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Foetal hyper-echogenic colon as an early sign of cystinuria

Nefrologia 2011;31(1):123-4
doi:10.3265/Nefrologia.pre2010.Sep.10636

To the Editor,

Cystinuria is a hereditary disease caused by a defect in the renal and intestinal tubular transport affecting cystine and the dibasic amino acids (lysine, ornithine and arginine).[1] It is transmitted as an autosomal recessive disorder and has a prevalence of about 1 in 7,000 live births, with a wide geographical variation and no predominance of sex. The clinical manifestations are effectively nephrolithiasis and its consequences (colic, haematuria, etc.) which usually occur in the second or third decades of life, although they can appear as early as the first year. It is the cause of 6%-10% of paediatric urolithiasis cases.[2] The cystine stone formation is due to the excessive concentration of this amino acid in urine and its high insolubility, especially when the urine is acidic.

We had the opportunity of studying a child, currently three years old, who was referred by his paediatrician when he was five months old, after an episode of gross haematuria, which revealed the presence of a stone in the nappy. It was the first child of non-consanguineous parents, without any previous significant pathology, but with a history of renal colic on the paternal side of the family. Ultrasound foetal studies during pregnancy revealed a colon hyperechogenicity without other intestinal abnormalities (Figures 1 and 2), and a slightly increased nuchal luminescence, with no other findings of interest. As a result, a sweat test was performed at birth to rule out cystic fibrosis and the result was normal.

Subsequent ultrasound images revealed multiple bilateral stones, which grew to a diameter of 1.4cm. Persistently high cystine elimination was detected in the urine (maximum 656mg/g creatinine at 7 months old). The renal glomerular function is normal (serum creatinine 0.28mg/dl), although there was a defect in the ability to concentrate (689mOsm/kg) and elevated urinary excretion of microalbumin (microalbumin/creatinine ratio 33.9µg/µmol).

During its evolution, numerous small stones have been expelled (over 50

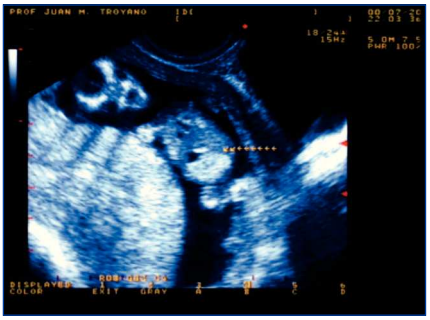

Figure 1. Hyperechogenic intestine with sound density similar to foetal bone

during the first year of life, measuring few mm in diameter), and the condition is otherwise asymptomatic. The weight-to-height ratio and psychomotor development during growth was normal. Pharmacological and dietary treatment with potassium citrate, captopril and D-penicillamine is currently being administered.

This is an early clinical presentation of cystinuria, reflecting the high lithogenic capacity of this condition. The particularity of the case is that the prenatal ultrasound found hyperechogenicity of the colon secondary to cystine crystal deposition. This form of presentation of cystinuria was described in 2006[3] and was subsequently confirmed.[4] The explanation for this finding is that the cystine crystals are formed in the foetal kidney, they enter the amniotic fluid and are then swallowed. The ultrasound finding of the foetal hyperechogenic colon has been traditionally related to cystic fibrosis,

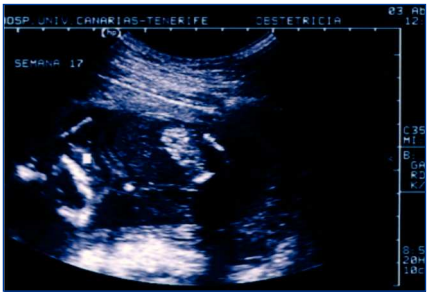

Figure 2. A similar situation early in the second trimester

which was why the studies needed to rule out the disease were performed at birth. The negative result and early clinical symptoms led to the diagnosis. Knowledge of this association may facilitate an early diagnosis of the disease, thus establishing an appropriate treatment.

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Acute renal failure as a presentation of an aortocaval fistula associated with abdominal aortic aneurism

Nefrologia 2011;31(1):124-6
doi:10.3265/Nefrologia.pre2010.Sep.10634

To the Editor,

Aortocaval fistula (ACF) is an uncommon complication of abdominal aortic aneurysms (AAA) that requires urgent action. However, they are diagnosed in only half of the cases, which increases postoperative mortality. Indicative of this condition are a continuous abdominal murmur and the presence of high-output heart failure (HF) or regional venous hypertension. Acute renal failure (ARF) may be the presentation of the ACF, as in the case described.

This is the case of a 71-year-old patient with a history of dyslipidaemia, hypertension, truncal obesity and coronary artery bypass. The patient was transferred to our hospital from the emergency department of another hospital due to anuric ARF unresponsive to dopamine or furosemide. The patient complained of self-limited sweating episodes, and had malaise, agitation, disorientation, and tachypnoea, blood pressure at 108/62mm Hg, a pulse of 91 beats/min and central venous pressure (CVP) of 30cm H_2O. An examination revealed bibasilar crackles and painful, enlarged liver, without a pulsatile abdominal mass or ankle oedema. Laboratory tests revealed the following data: urea 108mg/dl, creatinine 4.05mg/dl, pH 7.19 and bicarbonate 10.2mmol/l, with blood count and coagulation normal.

The chest x-ray showed a vascular redistribution pattern with small bilateral pleural effusion. A renal Doppler ultrasound showed normal-sized kidneys without signs of obstruction, a dilated right renal vein, and a large AAA. The CT scan with contrast showed an infrarenal, 10cm AAA with atherosclerosis and thrombosis, without retroperitoneal haematoma, as well as the flow of contrast to the vena cava and right renal vein during the arterial phase and the ACF (Figure 1).

With the diagnosis of ARF and CHF secondary to ACF, haemodialysis and surgery were performed, with control of the aneurysm neck, which required the ligation of left renal vein, the opening the aneurysmal sac (with the loss of 2 litres of blood), the closure of the ACF and placement of aorto-aortic bypass. The immediate postoperative diuresis was 70ml/h, but the patient developed refractory multiorgan failure and died three days after surgery.

The rupture of an aortic aneurysm and into other organs is uncommon. The inferior vena cava is the most common, followed by the iliac veins, the left renal vein and intestine.[1,2-4] Over 80% of ACF cases are due to the rupture of an **a**therosclerotic aortic aneurysm (average size 11cm),[2-5] with other causes being penetrating abdominal trauma; iatrogenia, mainly lumbar disc surgery or catheterisation; and occasionally others.[1,3]

Between 31% and 76% of the ACFs are detected during surgery after evacuating the clot from the aneurysm sac, which causes massive

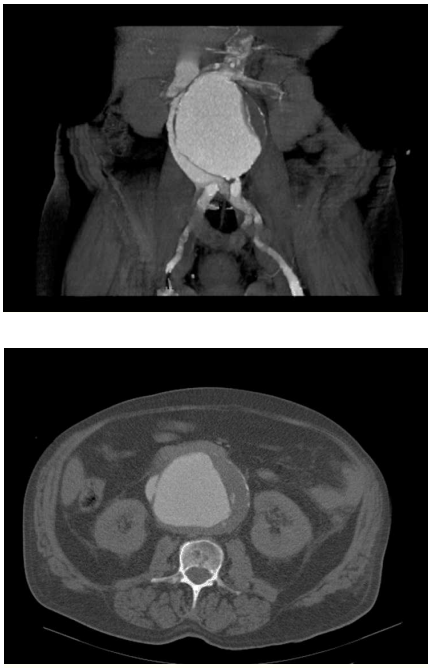

Figure 1. CT scan with contrast. Abdominal aorta aneurism and aortocaval fistula